**UNITED STATES DISTRICT COURT**
**EASTERN DISTRICT OF PENNSYLVANIA**

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| IN RE: NATIONAL FOOTBALL LEAGUE PLAYERS' CONCUSSION INJURY LITIGATION | No. 2:12-md-02323-AB<br>MDL No. 2323 |
| Kevin Turner and Shawn Wooden, *on behalf of themselves and others similarly situated*,<br><br>Plaintiffs,<br><br>v.<br><br>National Football League and NFL Properties, LLC, successor-in-interest to NFL Properties, Inc.,<br><br>Defendants. | Civil Action No. 2:14-cv-00029-AB |
| THIS DOCUMENT RELATES TO: ALL ACTIONS | |

**DECLARATION OF SAM GANDY, M.D., PH.D.**

Sam Gandy, M.D., Ph.D., affirms under penalty of perjury the truth of the following facts:

1. I am the Mount Sinai Professor of Alzheimer's Disease Research, Professor of Neurology and Psychiatry, Associate Director of the Mount Sinai Alzheimer's Disease Research Center in New York City, and Chairman Emeritus of the National Medical and Scientific Advisory Council of the Alzheimer's Association.

2. My complete *curriculum vitae* is attached at Exhibit 1. Experience and training of particular relevance to this declaration includes certification by the American Board of Psychiatry and Neurology as a Diplomate in the specialty of Neurology and by my nomination and service as Official Delegate from the American Academy of Neurology as author and quality

assessor of items for the creation of new certification in Brain Injury Science by the American Board of Physical Medicine and Rehabilitation.

3. I have reviewed generally the Class Action Settlement Agreement as of June 25, 2014, together with its exhibits (the "Settlement"), filed in the above captioned proceeding, with particular attention to Exhibits 1 and 2 of the Settlement.

4. Pathologically, CTE involves build-up of phosphorylated tau protein in the brain. Higher levels of tau build-up are believed to associate with more advanced stages of CTE. CTE is the only neurodegenerative disease that has been linked to a specific acquired cause – repeated head trauma. What sets CTE apart from other neurocognitive injuries is a relentlessly progressive course leading to a syndrome of psychological, mood, cognitive, and/or motor deficits that continue to progress even in the absence of further head trauma.

5. The primary clinical features of CTE include impairment of cognition, mood, behavior and/or movement. Individuals with neuropathologically confirmed CTE have significant problems with mood, behavior, and/or movement and not just problems with cognition. These behavioral, mood, and movement disorders are serious and devastating; they are equally as important and can be equally as disabling as the cognitive disorders that can result from head impacts.

6. For example, these mood and behavioral symptoms include impairment of executive function, poor impulse control, socially inappropriate, avolitional, and apathetic behaviors. Damage to the orbitofrontal regions of the brain can result in significant personality changes, including apathy, impulsivity, aggression, and the "short fuse" explosive behaviors that are typical of CTE as the illness is known based on neuropathological indexing. Such personality changes are consistent with the atrophy and other neuropathological changes of the

frontal lobes that have been described in nearly all reported cases of CTE. These mood and behavioral symptoms can have a devastating impact on an individual's life.

7. These mood and behavioral symptoms of CTE typically present in mid-life after a latency period – as long as years or decades after the exposure. Because CTE symptoms present much earlier than the symptoms of other neurodegenerative diseases, individuals with CTE face decades of disability, a challenge that others afflicted with neurodegenerative disease do not face.

8. As CTE progresses, individuals with CTE develop worsening memory impairment, language problems, motor dysfunction, and continued aggression. Dementia is evident in most individuals with CTE who survive to age 65.

9. Some patients with CTE, however, may never reach dementia. The high rates of suicides, accidents, and drug overdoses often lead to death before the individual reaches age 65. Thus, many persons with neuropathologically confirmed CTE do not have dementia at time of death.

10. Other cases of CTE may never advance past the mood and behavioral changes that are typical of how CTE first presents, at least in CTE as identified in neuropathological series. Even for those individuals whose CTE does not progress to dementia, the impact of CTE on a patient's ability to regulate his mood and behavior prior to ever reaching dementia can be devastating and totally incapacitating. For example, based on statements by family members that are published in the public domain, it is not clear that either Junior Seau and Dave Duerson would have qualified for dementia payments under the settlement. Both are former NFL players who displayed hallmark characteristics of CTE's mood and behavioral symptoms – hopelessness,

aggression, and poor impulse control. Both eventually committed suicide and were found to have CTE.

11. The Settlement does not compensate these mood, behavioral, or motor symptoms of CTE.

12. Although a definitive diagnosis of CTE in the living is currently beyond the reach of present medical technology, there are recommended diagnostic protocols for individuals who may have CTE. That recommended assessment includes neuropsychological evaluation, neurological examination, brain imaging, and blood and CSF biomarkers. Particular attention should be paid to cognitive function, mood, personality, behavior, and olfaction. The Settlement's testing protocol, however, does not meet this recommendation. It lacks neurological examination, brain imaging, and blood and CSF biomarker testing. It focuses only on cognitive function, not mood, personality, behavior, and olfaction.

13. Recent developments in medical diagnostic imaging technology, moreover, are moving toward giving physicians the ability to detect and diagnose CTE in living people. For example, PET tracers are available that bind to tau protein in the brain. Those tracers can then be highlighted using standard imaging technology, such as a PET scan. Combining those tau tracers with beta amyloid tracers can enable the clinician to distinguish between CTE and Alzheimer's. An Alzheimer's patient will show build-up of both substances, while most CTE patients will show only tau build-up.

14. Indeed, a research group in which I participated recently reported on the ability to use such PET tracers in a published, peer-reviewed paper. That study used both tau and beta amyloid tracers to study two individuals, one of whom was suspected of having Alzheimer's. The distribution of tau and beta amyloid, however, was more indicative of CTE than

Alzheimer's. The clinical misdiagnosis of CTE as Alzheimer's is not unusual. As research continues on these technologies, such tracers will become more sensitive, more accurate, and CTE diagnoses in living people will become more reliable.

15. CTE is a distinct, neurodegenerative disease. It is different from other neurodegenerative diseases, such as those that qualify for payment under the Settlement. For example, the neuropathology of a brain with Alzheimer's is different than that of a brain with CTE. Both brains show tau tangles but they differ in the frequency of presence of amyloid plaques. Yet all four of these diseases – Parkinson's, ALS, Alzheimer's, and CTE – can be definitively determined through examination of brain tissue on autopsy following death. Using currently approved technology, none of CTE, Alzheimer's, Parkinson's, or ALS can be definitively diagnosed during life.

16. Dementia is neither a single illness nor a single disease. Instead, it is a descriptor of a person's neurocognitive decline. Thus, some neurodegenerative diseases can lead to dementia. Alzheimer's, CTE, and Parkinson's, and ALS are all such diseases. The brain pathologies of these diseases begin well before any symptoms and well before the onset of dementia. Only after the disease has destroyed enough brain tissue in clinically important brain regions do the symptoms of dementia begin to present. Initial symptoms are not technically "dementia." Only when the disease has sufficiently progressed that a person's cognitive decline begins to interfere with independent functioning would an individual be characterized as having dementia.

Pursuant to 28 U.S.C. § 1746, I state under penalty of perjury that the foregoing is true and correct:

Sam Gandy, M.D., Ph.D.

Date: 09 Oct 2014

# EXHIBIT 1

April 6, 2014

# CURRICULUM VITAE

***Sam Gandy, M.D., Ph.D.***

## ACADEMIC APPOINTMENTS

| | |
|---|---|
| 2007- Present | Mount Sinai Professor of Alzheimer's Research<br>Professor of Neurology and Psychiatry (Dual Primary)<br>Icahn School of Medicine at Mount Sinai, New York, NY |
| 2001-2007 | Paul C. Brucker, M.D., Professor of Neuroscience<br>Professor of Neurology, Biochemistry and Molecular Biology<br>Founding Director, Farber Institute for the Neurosciences<br>Thomas Jefferson University, Philadelphia, PA |
| 1999-2000 | Raine Foundation Visiting Distinguished Professor<br>University of Western Australia, Perth WA, Australia |
| 1997-2001 | Research Scientist<br>The Nathan S. Kline Institute for Psychiatric Research, Orangeburg, NY |
| 1997-2001 | Professor of Psychiatry and Cell Biology<br>New York University School of Medicine, New York, NY |
| 1997-Present | Adjunct Professor of Molecular and Cellular Neuroscience<br>The Rockefeller University, New York NY |
| 1993-97 | Associate Professor and Laboratory Director<br>Department of Neurology and Neuroscience<br>The New York Hospital Cornell Medical Center, New York, NY |
| 1993-97 | Adjunct Associate Professor<br>The Rockefeller University, New York, NY |
| 1992-93 | Assistant Professor and Laboratory Director<br>Department of Neurology and Neuroscience<br>The New York Hospital-Cornell Medical Center, New York, NY |
| 1991-92 | Assistant Professor, Laboratory of Molecular and Cellular Neuroscience<br>The Rockefeller University, New York, NY |

## HOSPITAL APPOINTMENTS

| | |
|---|---|
| 2011-Present | Director, Center for Cognitive Health and NFL Neurological Center<br>Mount Sinai Hospital, New York NY |
| 2007-Present | Attending Neurologist, Mount Sinai Hospital, New York, NY |
| 2007-Present | Attending Neurologist<br>James J. Peters Veterans Affairs Medical Center, Bronx, NY |
| 2001-2007 | Attending Neurologist<br>Thomas Jefferson University Hospital, Philadelphia, PA |

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|---|---|
| 1986-88: | Attending Neurologist, The New York Hospital, New York, NY<br>General Neurology Clinic and Consult Service |
| 1983-86: | Resident and Clinical Associate in Neurology<br>The New York Hospital-Cornell Medical Center, New York, NY |
| 1982-83: | Intern, Department of Medicine, Presbyterian Hospital<br>Visiting Clinical Fellow, College of Physicians and Surgeons<br>Columbia University, Columbia-Presbyterian Medical Center, New York, NY |

## EDUCATION

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|---|---|
| 1976: | B.S., *summa cum laude*, Charleston Southern University (Chemistry) |
| 1982: | M.D., Ph.D., Medical University of South Carolina (Molecular Cell Biology) |

## POSTDOCTORAL TRAINING

| | |
|---|---|
| 1982-83: | PGY 1 Intern, Columbia University College of Physicians and Surgeons,<br>Supervisor: John Bilizekian |
| 1983-86: | PGY 2-4 Resident in Neurology, Cornell University Medical College<br>Supervisor: Fred Plum |
| 1986-91: | Postdoctoral Research Associate, The Rockefeller University<br>Supervisor: Paul Greengard |

## CERTIFICATION

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|---|---|
| 1988 | Diplomate in Neurology, American Board of Psychiatry and Neurology |

## LICENSURE

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|---|---|
| 7/1/1983 | New York, License # 154552 |
| 2/8/2002 | Pennsylvania, License # MD418573 |
| 7/13/2007 | Georgia, License # 059726 |

## HONORS/AWARDS/PATENTS

| | |
|---|---|
| 1976 | B.S., *summa cum laude* |
| 1981 | Alpha Omega Alpha |
| 2008 | Arthur Cherkin Memorial Award in Geriatric Medicine<br>University of California, Los Angeles |

### *Issued Patents*

| | |
|---|---|
| 5,385,915 | Treatment of amyloidosis associated with Alzheimer disease using modulators of protein phosphorylation<br>Issued: January 31, 1995 |
| 5,348,963 | Method of screening for modulators of amyloid formation<br>Issued: September 20, 1994 |

5,242,932 Treatment of amyloidosis associated with Alzheimer disease
Issued: September 7, 1993

4,874,694 Use of phosphoprotein patterns for diagnosis of neurological and psychiatric disorders; Issued: October 17,1989

## OTHER PROFESSIONAL APPOINTMENTS

### 1. Committee Memberships

a. Regional and State:
Ad Hoc Pilot Proposal Reviewer, Alzheimer Disease Core Center,
New York University, 1991-2000
b. Institutional:
Appointments and Promotions, Thomas Jefferson University
Committee on Special Awards, Mount Sinai School of Medicine

### 2. Current consultancies

Baxter Pharmaceuticals
Amicus Therapeutics
Janssen/Pfizer Alzheimer's Initiative
Diagenic

### 3. Editorships and Editorial Boards

**Present**

Associate Editor, *Alzheimer's Disease and Associated Disorders*, 1992-present
Associate Editor, *Molecular Neurodegeneration*, 2005-present

Editorial Advisory Board, *Neurodegenerative Diseases*, 2003-present
Editorial Board, *Journal of Neuroinflammation*, 2004-present
Editorial Board, *Public Library of Science: Medicine*, 2007-present
Member, Faculty of 1000 Biology, 2008-present
Editorial board, *The Journal of Biological Chemistry,* 2012-present

**Past**

Consulting Editor, *The Journal of Clinical Investigation*, 2003-2013

## ADMINISTRATIVE LEADERSHIP APPOINTMENTS

### INTERNAL

1992-1997 Designer, Neurology and Neuroscience Problem Based Curriculum
Weill Cornell Medical College

2001-2007 Founding Director, Farber Institute for Neurosciences
Founder, Alzheimer's Clinical Trials Program, Jefferson Medical College

2007-present Committee for Special Awards, Icahn School of Medicine at Mount Sinai
Friedman Brain Institute, Faculty Search Committee

2007-present Chief, Division of Neurodegeneration, Friedman Brain Institute

**EXTERNAL**

**National and International**

1993-2009 Ad Hoc IRG Member and Site Visitor, NINDS, NIA
1993-present Ad Hoc Reviewer, The Wellcome Trust

1995-2001 Member, NIH, Neurological Sciences-1 Initial Review Group
1997-1998 Chair, NIH, Neurological Sciences-1 Initial Review Group (Study Section)

2000-2006 Chair, Rotary Club CART Grant Award Committee
2001-2006 Chair, Scientific Advisory Board, Elizabeth and Zachary Fisher Foundation for Alzheimer's Research
2005-2009 Chair, Alzheimer's Association National Medical and Scientific Advisory Council

**STUDENT TRAINING RECORD**

| **NAME** | **LEVEL OF TRAINEE** | **ROLE IN TRAINING** | **TRAINING VENUE** | **TRAINEE'S CURRENT STATUS & INSTITUTION EMPLOYED** |
|---|---|---|---|---|
| Gregg Caporaso | Ph.D. Student | Direct Supervision | Laboratory | Asst. Prof. Neurology, NYU |
| Joseph Buxbaum | Postdoctoral Fellow | Direct Supervision | Laboratory | Professor of Psychiatry, Mt. Sinai, NYC |
| Kerstin Iverfeldt | Postdoctoral Fellow | Direct Supervision | Laboratory | Professor, Chair, Neurochemistry, Stockholm University |
| Toshiharu Suzuki | Postdoctoral Fellow | Direct Supervision | Laboratory | Professor, Hokkaido University |
| Christer Nordstedt | Postdoctoral Fellow | Direct Supervision | Laboratory | VP Neuroscience, Astra Zeneca, Sodertalje, Sweden |
| Huaxi Xu | Postdoctoral Fellow | Direct Supervision | Laboratory | Professor, Burnham Institute |
| Suzana Petanceska | Postdoctoral Fellow | Direct Supervision | Laboratory | Program Officer, NIA |

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| Parvathy Sarapavanavananthan (deceased) | Postdoctoral Fellow | Direct Supervision | Laboratory | Research Associate, UCSF, at the time of death |
| Ralph Martins | Postdoctoral Fellow | Direct Supervision | Laboratory | Professor, Edith Cowan University |
| Gunnar Gouras | Postdoctoral Fellow | Direct Supervision | Laboratory | Professor, Lund University |
| Jan Naslund | Postdoctoral Fellow | Direct Supervision | Laboratory | Staff Scientist, Astra Zeneca, Sodertalje, Sweden |
| Dun Sheng Yang | Postdoctoral Fellow | Direct Supervision | Laboratory | Research Associate, NKI |
| Jun Yao | Postdoctoral Fellow | Direct Supervision | Laboratory | Research Associate, Columbia University |
| Joshua Gatson | Postdoctoral Fellow | Direct Supervision | Laboratory | Postdoctoral Fellow, University of North Texas |
| Rachel Lane | Postdoctoral Fellow | Direct Supervision | Laboratory | Program Officer, ADDF |
| Soong Ho Kim | Postdoctoral Fellow | Direct Supervision | Laboratory | MSSM |
| Serene Keilani | Postdoctoral Fellow | Direct Supervision | Laboratory | Retired |
| Eugene Hone | Postdoctoral Fellow | Direct Supervision | Laboratory | Postdoctoral Fellow, Edith Cowan University |
| John Steele | Predoctoral Fellow | Direct Supervision | Laboratory | Postdoctoral Fellow, The Rockefeller University |
| Ina Caesar | Postdoctoral Fellow | Direct Supervision | Laboratory | Fellow, Linkoping University |
| Hannah Brautigam | Predoctoral Fellow | Direct Supervision | Laboratory | Undecided |

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|---|---|---|---|---|
| Elysse Knight | Postdoctoral Fellow | Direct Supervision | Laboratory | MSSM |

**DIDACTIC TEACHING ACTIVITIES**

| TEACHING ACTIVITY / TOPIC | LEVEL | ROLE | NUMBER OF LEARNERS | NUMBER OF HOURS PER WEEK / MONTH / YEAR | YEARS TAUGHT |
|---|---|---|---|---|---|
| Neurological Diagnosis | Medical School Course | Course Director and Lecturer | 100 | 5 hr/wk<br>9 mo/year | 1992-1995 |
| Problem Based Approach to Basic and Clinical Sciences | Medical School Course | Neurology and Neuroscience Section Head and PBL Leader | 100 | 5 hr/wk<br>9 mo/year | 1995-1997 |
| Molecular Basis of Neurological Disease Summer Course | Cold Spring Harbor Laboratory: Sub-specialty course | Course Director and Speaker | 25 | 18 hr/day<br>6 days/yr | 1996-2006 |
| Neuropsychiatric Research Course | Department of Psychiatry: Postgraduate Course | Course Director | 25 | 5 hr/wk<br>3 mo/year | 1997-2001 |
| Scientific Foundations of Clinical Medicine | Medical School Course | Course Director and Lecturer, Dementia Module | 100 | 5 hr/wk<br>3 mo/year | 2001-2007 |

| FUNDING SOURCE, PROJECT TITLE & NUMBER | ROLE IN PROJECT | DATES | DIRECT COSTS |
|---|---|---|---|
| NINDS 5 K08 NS001095<br>Characterization of a Neuron-specific phosphoprotein | PI | 7/1/86-6/30/91 | $80,000/Yr<br>5 Years |
| NIA Pilot Project<br>Neuron-Specific Phosphoproteins in Alzheimer CSF | PI | 1990-1992 | $50,000/Yr<br>2 Years |
| NIA 5 P01 AG010491<br>Interdisciplinary Approach to Alzheimer Drug Discovery | Program Co-Director | 1991-1996 | $900,000/Yr |
| NIA 5 P01 AG010491<br>Cell Biology of Amyloid Precursor Protein Processing in vitro, in vivo | Project Leader | 1991-1996 | $150,000/Yr |
| NIA 5 R01 AG011508<br>Molecular Cell Biology of Alzheimer Amyloidogenesis | PI | 1992-1997 | $120,000/Yr |
| NIA Pilot Project<br>Leadership & Excellence in Alzheimer's Disease | PI | 1993-1994 | $50,000/Yr |
| NIA ADRC P50 AG08702<br>Signal Transduction and Amyloid in Alzheimer's Disease | Co-Project Leader | 1994-1995 | $120,000/Yr |
| NIA R01 AG013780<br>Regulated Cleavage of Amyloid Precursor: Molecular Basis | PI | 1996-2001 | $150,000/Yr |
| NIA 5 P01 AG009464<br>Signal Transduction and Alzheimer's Disease – Cell Biological Studies | Deputy Program Director | 1990-2001<br>The Rockefeller University | $900,000/Yr |
| NIA 5 P01 AG009464<br>Cell Biological Studies of Amyloid Precursor Protein | Project Leader | | $150,000/Yr |
| NIA 5R01AG018237<br>Neuroanatomy of $GABA_A$ receptors in Alzheimer's Disease | PI | 2002-2005 | $120,000/Yr |
| NIA 5R01AG008206<br>Neurotransmitter Anatomy in Alzheimer's Disease | PI | 2002-2005 | $120,000/Yr |
| NINDS R01 NS41017<br>"Estrogen Modulation of Brain Abeta Metabolism *in vivo*" | PI | 2000-2007 | $706,500 |
| Cure Alzheimer's Fund<br>"Mouse Model of Intraneuronal and Vascular Abeta Oligomers" | PI | 11/01/07 - 10/31/09 | $100,000 |
| NIA R01 AG023611<br>"Presenilin Domains and Reconstitution of Catalysis" | PI | 7/1/05 - 06/30/10 | $828,833 |

| **FUNDING SOURCE, PROJECT TITLE & NUMBER** | **ROLE IN PROJECT** | **DATES** | **DIRECT COSTS/YR** | **SUPPLEMENTAL INFO** |
|---|---|---|---|---|
| NIA P01 AG010491 "Interdisciplinary Approach to Alzheimer Drug Discovery" | Director | 9/30/05-8/31/12 | $977,663 | Active |
| Cure Alzheimer's Fund "SorCS1, Diabetes, and Alzheimer's" | PI | 4/1/11-3/31/12 | $100,000 | Active; renewable |
| Amicus Pharmaceuticals | P! | 9/1/10-12/31/12 | $100,000 | Active, renewable |
| VA MERIT "Mouse Model of Intraneuronal Amyloid Beta Oligomerization" | PI | 7/1/10 - 6/30/13 | $175,000 | Active; renewable |
| NIA P50 AG005138 "Alzheimer's Disease Research Center" | Associate Director | 5/1/97-3/31/15 | $200,381 | Active; renewable |
| NINDS R01 "SorCS1, Diabetes, and Alzheimer's" | PI | 1/1/12-12/31/15 | $1,200,000 | Active; renewable |
| NIA R21 "Generation of Alzheimer's Brain Cells" | PI | 7/1/12 - 6/30/14 | $175,000 | Active; renewable |
| Cure Alzheimer's Fund "Foundation Grant for CAF Stem Cell Consortium" | PI | 3/1/13 - 2/28/14 | $100,000 | Active; renewable |
| Baxter Pharmaceuticals "Effect of Gammagard Liquid on Oligomer-Only Mouse Model" | PI | 7/1/12 - 6/30/14 | $200,000 | Active; renewable |

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| Louis B. Mayer Foundation | PI | 3/1/12 - 12/31/13 | $25,000 | Active; renewable |
| Constellation Wines | PI | 3/1/12 - 2/28/14 | $230,000 | Active; renewable |
| NIA R01 “Integrative Approach to Alzheimer’s Disease Complexity” | Multi PI | 09/01/13 - 08/31/18 | $200,000 | Active; renewable |
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**PUBLICATIONS**

**Peer Reviewed Original Contributions**

1. Bonnette, A.K. and ***Gandy***, S. Isotopic exchange in Prussian blue. J. Chemical Education 1981; 58:355-357.

2. Crouch, R.K., ***Gandy***, S., Kimsey, G., Galbraith R.A., Galbraith, G.M. and Buse, M.G. The inhibition of islet superoxide dismutase by diabetogenic drugs. Diabetes 1981; 30: 235-241.

3. ***Gandy***, S.E., Buse, M.G. and Crouch, R.K. Protective role of superoxide dismutase against the beta cell toxicity of diabetogenic drugs in rats and isolated canine islets. J. Clin. Invest. 1982; 70: 650-658.

4. ***Gandy***, S.E., Buse, M.G., Sorenson, J.R.J. and Crouch, R.K. Attenuation of streptozotocin diabetes with superoxide dismutase-like copper-(II)-(diisopropylsalicylate)2 in the rat. Diabetologia 1983; 24: 437-440.

5. Murray, G.J., Youle, R.J., ***Gandy***, S.E., Zirzow, G.C. and Barranger, J.A. Purification of ß-glucocerebrosidase by preparative scale HPLC: The use of ethylene glycol containing buffers for chromatography of hydrophobic glycoprotein enzymes. Anal. Biochem. 1984; 147:301-310.

6. Crouch, R.K., ***Gandy***, S.E., Patrick, J., Reynolds, S., Buse, M.G. and Simson, J.A. Localization of copper- zinc superoxide dismutase in the endocrine pancreas. Exp. and Molec. Pathol. 1984; 41: 377-383.

7. ***Gandy***, S.E., Snow, R.B., Zimmerman, R.D. and Deck, M.D.F. Cranial nuclear magnetic resonance imaging in head trauma. Ann. Neurol. 1984; 16:254-257.

8. Snow, R.B., Zimmerman, R.D., ***Gandy***, S.E. and Deck, M.D.F. Comparison of MRI and computed tomography in the evaluation of head injury. Neurosurgery 1986; 18:45-52.

9. ***Gandy***, S.E. and Payne, R. Back pain in the elderly: updated diagnosis and management. Geriatrics 1986; 41(12): 59-62, 67-74.

10. Goldman, S.A. and ***Gandy***, S.E. Squamous carcinoma as a late complication of intracerebroventricular epidermoid. J. Neurosurg. 1987; 66: 618-620.

11. ***Gandy***, S.E. and Heier, L.A. Clinical features and magnetic resonance images of primary intracranial arachnoid cysts. Ann. Neurol. 1987; 21:342-348.

12. Feldmann, E., ***Gandy***, S.E., Becker, R., Zimmerman, R., Thaler, H.T., Posner, J.B. and Plum, F. Magnetic resonance imaging demonstrates descending transtentorial herniation. Neurology 1988; 38: 697-701.

13. ***Gandy***, S., Czernik, A., and Greengard, P. Phosphorylation of Alzheimer disease amyloid precursor peptide by protein kinase C and Ca+2/calmodulin-dependent protein

kinase II. Proc. Natl. Acad. Sci. USA 1988; 85: 6218-6221.

14. Buxbaum, J.D., ***Gandy***, S.E., Cicchetti, P., Ehrlich, M.E., Czernik, A.J., Fracasso, P., Ramabhadran, T.V., Unterbeck, A.J., and Greengard, P. Processing of Alzheimer ß/A4 amyloid precursor protein: Modulation by agents that regulate protein phosphorylation. Proc. Natl. Acad. Sci. USA 1990; 87:6003-6006.

15. ***Gandy***, S.E., Grebb, J.A., Rosen, N.L., Albert, K.A., Devinsky, O., Blumberg, H., Anderson, M.B., Cedarbaum, J.M., Porter, R.J., Sedvall, G., Posner, J.B. and Greengard, P. General assay for phosphoproteins in CSF: A candidate marker for paraneoplastic cerebellar degeneration. Annals of Neurology 1990; 28: 829-833.

16. Cedarbaum, J.M., ***Gandy***, S.E. and McDowell, F.H. "Early" initiation of levodopa treatment does not promote the development of motor response fluctuations, dyskinesias or dementia in Parkinson's disease. Neurology 1991; 41: 622-629.

17. Nordstedt, C., ***Gandy***, S.E., Alafuzoff, I., Caporaso, G.L., Iverfeldt, K., Grebb, J.A., Winblad, B. and Greengard, P. Alzheimer ß/A4 amyloid precursor protein in human brain: Aging-associated increases in holoprotein and proteolytic fragment. Proc. Natl. Acad. Sci. 1991; 88:8910-8914.

18. ***Gandy***, S., Bhasin, R., Ramabhadran, T., Koo, E., Price, D., Goldgaber, D., and Greengard, P. Alzheimer ß/A4 amyloid precursor protein: Evidence for putative amyloidogenic fragment. J. Neurochem. 1992; 58: 383-386.

19. Caporaso, G., ***Gandy***, S., Buxbaum, J., and Greengard, P. Chloroquine inhibits intracellular degradation but not secretion of Alzheimer ß/A4 amyloid precursor protein. Proc. Natl. Acad. Sci. U.S.A. 1992; 89: 2252-2256.

20. Caporaso, G., ***Gandy***, S., Buxbaum, J., Ramabhadran, T., and Greengard, P. Protein phosphorylation regulates secretion of Alzheimer ß/A4 amyloid precursor protein. Proc. Natl. Acad. Sci. U.S.A. 1992; 89, 3055-3059.

21. Suzuki, T., Nairn, A., ***Gandy***, S., and Greengard, P. Phosphorylation of Alzheimer amyloid precursor protein by protein kinase C. Neuroscience 1992; 48:755-761.

22. Buxbaum, J., Oishi, M., Chen, H., Pinkas-Kramarski, R., Jaffe, E., ***Gandy***, S., and Greengard, P. (1992) Cholinergic agonists and interleukin 1 regulate processing and secretion of the Alzheimer ß/A4 amyloid protein precursor. Proc. Natl. Acad. Sci. U.S.A. 1992; 89:10075-10078.

23. Nordstedt, C., Caporaso, G., Thyberg, J., ***Gandy***, S., and Greengard, P. Identification of Alzheimer ß/A4 amyloid precursor protein in clathrin coated vesicles purified from PC12 cells. J. Biol. Chem. 1993; 268:608-612.

24. Ramabhadran, T., ***Gandy***, S., Ghiso, J., Czernik, A., Ferris, D., Bhasin, R., Goldgaber, D., Frangione, B., and Greengard, P. Proteolytic processing of human amyloid ß protein precursor in insect cells: Major carboxyl terminal fragment is identical to its human counterpart. J. Biol. Chem. 1993; 268:2009-2012.

25. Knops, J., ***Gandy***, S., Greengard, P., Lieberburg, I., and Sinha, S. Serine phosphorylation of the secreted extracellular domain of APP. Biochem. Biophys. Res. Commun. 1993; 197: 380-385.

26. da Cruz e Silva, O., Iverfeldt, K., Oltersdorf, T., Sinha, S., Lieberburg, I., Ramabhadran, T., Suzuki, T., Sisodia, S., ***Gandy***, S., Greengard, P. Regulated cleavage of Alzheimer ß-amyloid precursor protein in the absence of the cytoplasmic tail. Neuroscience 1993; 57: 873-877.

27. Caporaso, G., Takei, K., ***Gandy***, S., Matteoli, M., Mundigl, O., Greengard, P., de Camilli, P. Morphologic and biochemical analysis of the intracellular trafficking of the Alzheimer ß/A4 amyloid precursor protein. J. Neuroscience 1994; 14:3122-3138.

28. Nordstedt, C., Naslund, J., Thyberg, J., Messamore, E., ***Gandy***, S., Terenius, L. Human neutrophil phagocytic granules contain a truncated, soluble form of the Alzheimer ß/A4 amyloid precursor protein. J. Biol. Chem. 1994; 269:9805-9810.

29. Cheung, T.T., Ghiso, J., Shoji, M., Cai, X.-D., Golde, T., ***Gandy***, S., Frangione, B., Younkin, S. Characterization by radiosequencing of the carboxyl-terminal derivatives produced from normal and mutant amyloid ß protein precursors. Amyloid 1994; 1:30-38.

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